and his colleagues say, larger studies are necessary to assess the effects of patient information leaflets. We still will not know, however, which patients will be alarmed and which reassured by a knowledge of the side effects of their treatment, nor can we predict the effect on compliance. The most we can say is that patients have a right to this information and having better informed patients usually leads to more useful discussions between doctor and patient. We should favour a more open approach but also ask for more accurate data on which to base the information that we provide.

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Goats' milk for infants and children

Feeding goats' milk to babies is becoming increasingly popular and so requires more careful consideration than in the past. The main benefit claimed is that it is less allergenic than cows' milk and is a suitable substitute for babies who are allergic to the latter. Until recently goats' milk was readily available only in health food shops. Now, however, raw milk is sold in many parts of Britain and a spray dried powdered feed is being imported from New Zealand. A brochure produced by the manufacturers (Healtheries) of the powdered feed makes substantial claims[1]: "Goats' milk is especially recommended to those who suffer from allergies to cows' milk and other staple foods and stomach ulcers. It is important for fretful babies, the elderly and those suffering from nervous indigestion, insomnia and rheumatism.... It is said to be unlike cows' milk in that it does not form excess mucus."

These general claims would be probably harmless were it not for the repeated suggestion that goats' milk is suitable for infant feeds. The claims made for its benefits in "allergy" are an echo of the current debate in medical circles and are probably no more far fetched than some professional views. Children (as opposed to babies under 6 months) with genuine or supposed food allergies are unlikely to suffer great harm if goats' milk is added to their diet and so to the list of true, possible, or imagined allergens.

Potentially much more serious is the advice that goats' milk is a suitable substitute for conventional milk feeds when babies have supposed intolerance or allergy to cows' milk protein. A pamphlet produced by the British Goat Society recommends the use of raw milk "without boiling or pasteurisation" provided that the "hygiene of production of the milk is satisfactory."[2] This advice is dangerous. No untreated milk should ever be fed to young babies because of the risk of bacterial infection. By a peculiar anomaly goats' milk does not come under the government regulations that apply to most foods—for example, compulsory pasteurisation orders apply only to cows' milk.

The spray dried powder is less likely to carry any risk of infection. Unfortunately its composition is unsatisfactory as an infant formula in several ways. Its solute load is high—the content of sodium, potassium, and other electrolytes is similar to that of cows' milk. It also has a similar protein content to cows' milk, so that the production of urea and therefore the concentrations of urea in the blood and urine of babies fed goats' milk may be predicted to be of the same order as in those of babies fed unmodified cows' milk. Thus the use of goats' milk powder for infant feeding carries the same risk, predisposing to hypertonic dehydration, as does cows' milk powder.[3-5] Goats' milk is deficient in folic acid and (probably) vitamin B_{12}. It may also be deficient in vitamins C and D. The calcium and phosphorus ratios of cows' milk and goats' milk are similar. The risk of hypocalcaemic tetany in neonates is therefore the same for both.

Despite all the current interest goats' milk is scantily mentioned in medical publications. Since 1977 little research into its use has been reported, and certainly no evidence has appeared to support the many claims made for it, particularly not for its value to allergic infants and children.[6-9] No worthwhile evidence exists to justify giving goats' milk to young children. Indeed, feeding unmodified goats' milk, whether raw or powdered, to infants has all the disadvantages of giving them unmodified cows' milk. The DHSS report *Artificial Feeds for the Young Infant* considers goats' milk "unsuitable," and I can only agree with this view.[10]

If, however, despite the lack of nutritional or medical evidence of benefit parents choose to feed their babies with goats' milk they may avoid the main hazards by using the following guidelines. Firstly, raw goats' milk should be pasteurised or boiled. Animals should have been tested for both tuberculosis and brucellosis. The present loophole in the laws on milk hygiene should be closed without delay in view of the apparent growing popularity of goats' milk. Secondly, because of its high solute content, goats' milk should be diluted to threequarter strength. This decreases its energy content and some form of carbohydrate such as sucrose should be added. Undiluted goats' milk is unsafe for babies under 6 months of age, and even diluted is not ideal. Thirdly, supplements of folic acid and vitamin B_{12} are needed in addition to the supplement of vitamins A, C, and D given to babies fed cows' milk formulas.

Should goats' milk ever be recommended on medical grounds the ideal would be a prepared infant formula meeting the requirements of the DHSS report.[10] No such formula is currently available, but in view of the evidence it is questionable whether resources should be used to produce one.

Finally, any goats' milk product should have a stated shelf

life, which needs to be confirmed by testing. This applies to dried, frozen, and fresh milk. It should be labelled as unsuitable for babies under 6 months of age; and recommendations about vitamin and folic acid supplements and a nutritional analysis should be available. The free availability of a product labelled as an "All purpose whole milk powder," without qualification, is particularly dangerous.

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Towards a medical eugenics?

Almost exactly a year ago the *BMJ* acknowledged the imminent arrival of the future in an editorial on the impact of the new genetics on the prenatal diagnosis of genetic disease.[1] The new genetics progresses less sedately than the old, and in the months that have elapsed since then many of the questions raised in general terms in that article have assumed a more concrete and immediate form. Peter Harper has already outlined[2] some of the implications of the newly discovered genetic marker for Huntington's disease[3]; a very similar marker was reported at about the same time for the much commoner phenylketonuria[4]; and on page 431 Pope *et al* describe the direct detection of one of the collagen gene abnormalities responsible for osteogenesis imperfecta.

Meanwhile, with improvements in techniques for sampling fetal tissue during the first trimester of pregnancy,[5 6] the risk associated with prenatal diagnosis has probably now dropped below the estimated 2-5% risk of a genetically defective child.[7] Plainly the future has its foot in the door, and some argue that it cannot be long before screening for genetic disease has to be considered seriously as a routine part of antenatal care.

Are we, then, on the brink of eliminating from the population all the genetic diseases human flesh is heir to? We are not; and some of the most serious limitations—as well as some of the most spectacular possibilities—of current molecular genetic technology are illustrated particularly clearly by the two papers on Huntington's disease[3] and osteogenesis imperfecta.

From the point of view of the new genetics the crucial difference between the two diseases is that in osteogenesis imperfecta the defective gene is known and can be directly detected, whereas in Huntington's disease the gene remains unknown and its inheritance can be detected only indirectly and not always reliably. Not that osteogenesis imperfecta is by any means perfectly understood; it embraces several distinct connective tissue diseases of varying severity, and in fact one of the interesting points to emerge from the molecular analysis by Pope and his colleagues is that at least the lethal form that they have investigated requires more than one mutant gene.

In all cases of osteogenesis imperfecta, however, there are mutations in one or other of the genes for collagen. When a genetic disease results from a defect in such an abundant protein it is now relatively simple to make a radiolabelled DNA copy of the messenger RNA specifying the protein; the DNA copy will by its chemical nature adhere to the original gene in preparations of human DNA and so can be used as a probe to detect it. Whether the probe will distinguish a mutant from a normal gene depends on the vagaries of the battery of bacterial enzymes used by new geneticists to cleave the three million or so base pairs of human DNA into gene sized and below gene sized fragments. The bacterial enzymes will cut the DNA only at certain preferred sites. If one of those sites is affected by a mutation then the mutant gene will be cleaved into fragments of a different size from those of the normal gene. From this point of view Pope and his colleagues, investigating four cases of lethal osteogenesis imperfecta, were lucky: an alpha 1(I) collagen gene in their patients proved to have a large deletion easily detected by the bacterial enzymes (which are known technically as restriction enzymes—hence the term restriction fragments for the pieces into which they cleave DNA).

Because of this relatively gross defect Pope *et al* can offer absolutely reliable prenatal detection of the mutant gene; but they cannot on that basis alone predict lethal osteogenesis imperfecta. In each of their four cases only one of the parents had the mutant collagen gene; no abnormality could be detected in the other parent. But the parents carrying the mutant gene were either phenotypically normal or very mildly affected, and the other parents were normal. Thus the identified mutant collagen gene must be interacting with another, unknown gene (Pope *et al* guess that it is another collagen gene) to produce the lethal disease. Since they are unable to identify the second gene half of those fetuses diagnosed on the basis of the known mutant alone will presumably be normal. Conversely, half of those normal fetuses not carrying the known mutation will presumably be carriers for the unknown one.

All of this is in stark contrast to Huntington's disease, in which there is no such thing as symptomless carrier state and anyone carrying the gene will sooner or later develop the disease. Because the gene is unknown, however, even with the new marker it will be impossible to identify all individuals carrying it. Peter Harper has already discussed the distinctive problems posed by the late onset and ghastly prognosis of the disease; but it is worth spelling out what, in those special circumstances, can and cannot be expected of a genetic marker.

The marker for Huntington's disease is simply a piece of anonymous DNA (named G8) that happens to lie close enough to the Huntington's disease gene to be reliably inherited with it. The value of the G8 fragment as a genetic marker lies in its containing variations (polymorphisms) that are detectable by bacterial restriction enzymes and thus result in its being cleaved into fragments of different lengths in different people. Gusella *et al*, who discovered the marker, identified four variants (haplotypes) which they designated